Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	1	clckb	US-PGPUB; USPAT; DERWENT	ADJ	ON	2005/03/07 15:39
L2	1094	Clc and chloride	US-PGPUB; USPAT; DERWENT	ADJ	ON	2005/03/07 15:40
L3	56	Clc and chloride.ab.	US-PGPUB; USPAT; DERWENT	ADJ	ON	2005/03/07 15:42
L4	114	ClCK	US-PGPUB; USPAT; DERWENT	ADJ	ON	2005/03/07 15:42
L5	5	ClCK and chloride	US-PGPUB; USPAT; DERWENT	ADJ	ON	2005/03/07 15:43
L6	0	ClCK2b and chloride	US-PGPUB; USPAT; DERWENT	ADJ	ON	2005/03/07 15:43

1°= 10-15-03

File 5:Biosis Previews(R) 1969-2005/Feb W4
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Set Items Description
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Set	Items	Description
S1	4	CLCKB
S2	3	CHANNEL()KB
S3	274	AU='LANG FLORIAN' OR AU='LANG FLORIAN J W'
S4	67	AU='WALDEGGER SIEGFRIED' OR AU='WALDEGGER SIEGRIED'
S5	2	AU='LAMPERT ANGELIKA'
S6	51	S3 AND S4
S7	9	S6 AND CHLORIDE

? t s1/7/1-4

1/7/1
DIALOG(R)File 5:Biosis Previews(R)

0015143594 BIOSIS NO.: 200500050344
Novel mutations of the chloride channel Kb gene in two Japanese patients clinically diagnosed as Bartter syndrome with hypocalciuria
AUTHOR: Fukuyama Shigeru; Hiramatsu Misako; Akagi Motohiro; Higa Mutumi; Ohta Takao (Reprint)
AUTHOR ADDRESS: Fac MedDept Pediat, Univ Ryukyus, 207 Uehara, Okinawa, 9030125, Japan**Japan
AUTHOR E-MAIL ADDRESS: tohta@med.u-ryukyu.ac.jp
JOURNAL: Journal of Clinical Endocrinology & Metabolism 89 (11): p 5847-5850 November 2004 2004
MEDIUM: print
ISSN: 0021-972X _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Hypokalemic metabolic tubulopathy, such as in Bartter syndrome and Gitelman syndrome, is caused by the dysfunction of renal electrolyte transporters. Despite advances in molecular genetics with regard to hypokalemic metabolic tubulopathy, recent reports have suggested that the phenotype-genotype correlation is still confusing, especially in classic Bartter and Gitelman syndromes. We report here two Japanese patients who suffered from clinically diagnosed classic Bartter syndrome but who presented hypocalciuria. Hypocalciuria is generally believed to be a pathognomonic finding of NCCT malfunction. To better understand the genotype-phenotype correlation in these two cases, we screened four renal electrolyte transporter genes (Na-K-2Cl cotransporter (NKCC2), renal outer medullary K channel (ROMK), Cl channel Kb (%%%ClCKb%%%), and Na-Cl cotransporter (NCCT)) by the PCR direct sequencing method. We identified three ClC-Kb allelic variants, including two new mutations (L27R and W610X in patient 1 and a G to C substitution of a 3' splice site of intron 2 and W610X in patient 2). We did not find any mutations in the other three genes. Our present data suggest that some ClC-Kb mutations may affect calcium handling in renal tubular cells.

1/7/2
DIALOG(R)File 5:Biosis Previews(R)

0013223243 BIOSIS NO.: 200100395082
Bartter' syndrome type 3 (BS3): An unusual cause of nephrolithiasis
AUTHOR: Colussi G (Reprint); De Ferrari M E (Reprint); Bettinelli A; Tedeschi S; Cesareo L; Civati G (Reprint)
AUTHOR ADDRESS: Renal Unit, Riguarda-Ca' Granda Hospital, Milan, Italy** Italy
JOURNAL: Nephrology Dialysis Transplantation 16 (6): pA14 June, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Congress of the European Renal Association and the European Dialysis and Transplant Association Vienna, Austria June

24-27, 2001; 20010624
SPONSOR: European Renal Association
European Dialysis and Transplant Association
ISSN: 0931-0509
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

1/7/3
DIALOG(R)File 5:Biosis Previews(R)

0011959401 BIOSIS NO.: 199900219061
Expression of CLCN voltage-gated chloride channel genes in human blood vessels
AUTHOR: Lamb Fred S (Reprint); Clayton Gerald H; Liu Bei-Xing; Smith Roderic L; Barna Thomas J; Schutte Brian C
AUTHOR ADDRESS: Department of Pediatrics, University of Iowa Hospitals, 5040C RCP, Iowa City, IA, 52242, USA**USA
JOURNAL: Journal of Molecular and Cellular Cardiology 31 (3): p657-666 March, 1999 1999
MEDIUM: print
ISSN: 0022-2828
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Expression of CLCN Voltage-gated Chloride Channel Genes in Human Blood Vessels. Journal of Molecular and Cellular Cardiology (1999) 31, 657-666. Chloride (Cl) ion channels play a critical role in the response of both vascular smooth muscle (VSM) and endothelial (ENDO) cells to agonist stimulation. In VSM, agonist-induced Cl currents produce membrane depolarization, resulting in calcium influx through voltage-sensitive channels. ENDO cells also activate Cl currents after either agonist applicaiton or perturbation of cell volume. Although some of these currents have been characterized biophysically, the genes involved have not been identified. The CLCN family of voltage-dependent Cl channel genes comprises nine members (CLCN1-7, Ka and Kb) which demonstrate quite diverse functional characteristics while sharing significant sequence homology. We used Northern-blot analysis to study the expression of these Cl channel genes in cultured human aortic and coronary VSM cells and in aortic ENDO cells. CLCN3 is by far the most abundant CLC channel mRNA in both VSM and ENDO cells. Lower levels of expression are seen for CLCN2, CLCN4, CLCN5 and CLCN6. Expression levels were similar in VSM and ENDO cells except for CLCN4 which was more highly expressed in ENDO cells. In situ hybridization was used to confirm the expression of CLCN3 in intact human fetal lung. CLCN3 message was seen in VSM and ENDO cells of both large and small pulmonary vessels, indicating that their detection by Northern blotting was not an artifact of cell culture. CLCN3 is also expressed in pulmonary epithelial and bronchial smooth muscle cells but not in chondrocytes or pulmonary interstitial cells. Recent studies suggest that CLCN3 may encode the swelling-induced Cl conductance. We used whole cell patch clamp recording to demonstrate swelling-induced Cl currents in these cultured VSM cells. This suggests that the CLCN3 protein is expressed; however, the functional role of this current in VSM remains to be determined.

1/7/4
DIALOG(R)File 5:Biosis Previews(R)

0011858511 BIOSIS NO.: 199900118171
Gitelman syndrome due to mutation in the chloride channel %%%CLCKB%%%
AUTHOR: Jeck N; Konrad M; Reinalter S; Seyberth H W
AUTHOR ADDRESS: Philipps-Univ., Dep. Pediatr., Marburg, Germany**Germany
JOURNAL: Kidney and Blood Pressure Research 21 (2-4): p180 1998 1998
MEDIUM: print
CONFERENCE/MEETING: Congress of Nephrology 1998 Joint Scientific Meeting of the Society Nephrology Erlangen, Germany September 19-22, 1998; 19980919

SPONSOR: The Society for Nephrology
ISSN: 1420-4096
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

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2/7/1
DIALOG(R)File 5:Biosis Previews(R)

0015143594 BIOSIS NO.: 200500050344
Novel mutations of the chloride %%%channel%%% %%%Kb%%% gene in two Japanese patients clinically diagnosed as Bartter syndrome with hypocalciuria
AUTHOR: Fukuyama Shigeru; Hiramatsu Misako; Akagi Motohiro; Higa Mutumi; Ohta Takao (Reprint)
AUTHOR ADDRESS: Fac MedDept Pediat, Univ Ryukyus, 207 Uehara, Okinawa, 9030125, Japan**Japan
AUTHOR E-MAIL ADDRESS: tohta@med.u-ryukyu.ac.jp
JOURNAL: Journal of Clinical Endocrinology & Metabolism 89 (11): p 5847-5850 November 2004 2004
MEDIUM: print
ISSN: 0021-972X _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Hypokalemic metabolic tubulopathy, such as in Bartter syndrome and Gitelman syndrome, is caused by the dysfunction of renal electrolyte transporters. Despite advances in molecular genetics with regard to hypokalemic metabolic tubulopathy, recent reports have suggested that the phenotype-genotype correlation is still confusing, especially in classic Bartter and Gitelman syndromes. We report here two Japanese patients who suffered from clinically diagnosed classic Bartter syndrome but who presented hypocalciuria. Hypocalciuria is generally believed to be a pathognomonic finding of NCCT malfunction. To better understand the genotype-phenotype correlation in these two cases, we screened four renal electrolyte transporter genes (Na-K-2Cl cotransporter (NKCC2), renal outer medullary K channel (ROMK), Cl %%%channel%%% %%%Kb%%% (ClCKb), and Na-Cl cotransporter (NCCT)) by the PCR direct sequencing method. We identified three ClC-Kb allelic variants, including two new mutations (L27R and W610X in patient 1 and a G to C substitution of a 3' splice site of intron 2 and W610X in patient 2). We did not find any mutations in the other three genes. Our present data suggest that some ClC-Kb mutations may affect calcium handling in renal tubular cells.

2/7/2
DIALOG(R)File 5:Biosis Previews(R)

0014478721 BIOSIS NO.: 200300447440
Analysis of renal tubular electrolyte transporter genes in seven patients with hypokalemic metabolic alkalosis.
AUTHOR: Fukuyama Shigeru; Okudaira Shoko; Yamazato Syosin; Yamazato Masahiro; Ohta Takao (Reprint)
AUTHOR ADDRESS: Department of Pediatrics, Faculty of Medicine, University of the Ryukyus, 207 Uehara, Nishihara, Okinawa, 903-0125, Japan**Japan
AUTHOR E-MAIL ADDRESS: tohta@med.u-ryukyu.ac.jp
JOURNAL: Kidney International 64 (3): p808-816 September 2003 2003
MEDIUM: print
ISSN: 0085-2538 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: Disorders that manifest hypokalemic metabolic alkalosis, such as Bartter's syndrome and Gitelman's syndrome, are caused by the malfunction of renal tubular electrolyte transporters. Bartter's

syndrome may be linked to dysfunction of Na-K-2Cl cotransporter (NKCC2), renal outer medullary K channel (ROMK), or Cl %%%channel%%% %%%Kb%%% (ClC-Kb), while Gitelman's syndrome may be linked to Na-Cl cotransporter (NCCT) dysfunction. However, previous genetic analyses in these syndromes have included many heterozygotes for each gene and there has been no further analysis of other genes. Thus, to clarify the interaction of these transporter genes, in the present study we investigated all 4 transporter genes in 7 patients with hypokalemic metabolic alkalosis. Methods: Seven patients from 5 families (patients A-G) were collected, and a mutation analysis of the 4 renal electrolyte transporter genes was performed by direct sequencing. Results: We identified 12 mutations in these 7 patients. Three mutations (del245Y in NKCC2, R1009X in NCCT, V524I in ClC-Kb) have not been reported previously. In NKCC2 gene screening, patient A was homozygous for del245Y. In ClC-Kb gene screening, L27R was detected in patients B, D, and E. V524I was detected in patient C. Both T562M and E578K were observed in patients B and E. In NCCT gene screening, patients B-G shared a common novel mutation, R1009X, and patients D, E, F, and G carried this mutation in both alleles. Patients B and C carried R1009X in one allele, and a 6-amino acid insertion in exon 6 and L849H in another allele, respectively. The 4 other mutations did not result in any amino acid exchange. Despite the NCCT gene mutation, patients C and E showed normomagnesemia. Conclusion: Our findings demonstrate that in Bartter's and Gitelman's syndromes, it may not be uncommon to see mutations in several causative transporter genes.

2/7/3
DIALOG(R)File 5:Biosis Previews(R)

0008926577 BIOSIS NO.: 199396090993
Effect of KB-2796, a new diphenylpiperazine calcium antagonist, on voltage-dependent calcium currents and oxidative metabolism in dissociated mammalian CNS neurons
AUTHOR: Akaike Norio (Reprint); Ishibashi Hitoshi; Hara Hideaki; Oyama Yasuo; Ueha Toshiko
AUTHOR ADDRESS: Dep. Neurophysiol., Tohoku Univ. Sch. Med., Sendai 980, Japan**Japan
JOURNAL: Brain Research 619 (1-2): p263-270 1993
ISSN: 0006-8993
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The effects of KB-2796, 1-(bis(4-fluorophenyl)methyl)-4-(2,3,4-trimethoxybenzyl)piperazine-2HCl, on the low- and high-voltage activated Ca-2+ currents (LVA and HVA I-Ca, respectively) and on oxidative metabolism were studied in neurons freshly dissociated from Tat brain. KB-2796 reduced the peak amplitude of LVA I-Ca in a concentration-dependent manner with a threshold concentration of 10-7 M when the LVA I-Ca was elicited every 30 s in the external solution with 10 mM Ca-2+. The concentration for half-maximum inhibition (IC-50) was 1.9 times 10-6 M. At 10-5 M or more of KB-2796, a complete suppression of the LVA I-Ca was observed in the majority of neurons tested. There was no apparent effect on the current-voltage (I-V) relationship and the current kinetics. KB-2796 delayed the reactivation and enhanced the inactivation of the Ca-2+ channel for LVA I-Ca voltage- and time-dependently, suggesting that KB-2796 preferentially binds to the inactivated Ca-2+ %%%channel%%%. %%%KB%%%-2796 at a concentration of 3.0 times 10-6 M also decreased the peak amplitude of the HVA I-Ca without shifting the I-V relationship. In addition, KB-2796 reduced the oxidative metabolism (the formation of reactive oxygen species) of the neuron in a concentration-dependent manner with a threshold concentration of 3 times 10-6 M. It is suggested that the inhibitory action of KB-2796 on the neuronal Ca-2+ influx and the oxidative metabolism, in combination with a cerebral vasodilatory action, may reduce ischemic brain damage.

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7/3/1
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0015137205 BIOSIS NO.: 200500043955
Regulation of CLC-Ka/barttin by the ubiquitin ligase Nedd4-2 and the serum- and glucocorticoid-dependent kinases
AUTHOR: Embark Hamdy M; Boehmer Christoph; Palmada Monica; Rajamanickam Jeyaganesh; Wyatt Amanda W; Wallisch Sabine; Capasso Giovambattista; Waldegger Petra; Seyberth Hannsjoerg W; %%%Waldegger Siegfried%%%; %%%Lang Florian%%% (Reprint
AUTHOR ADDRESS: Inst PhysiolDept Physiol 1, Univ Tubingen, Gmelinstr 5, D-72076, Tubingen, Germany**Germany
AUTHOR E-MAIL ADDRESS: florian.lang@uni-tuebingen.de
JOURNAL: Kidney International 66 (5): p1918-1925 November 2004 2004
MEDIUM: print
ISSN: 0085-2538 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/2
DIALOG(R)File 5:Biosis Previews(R)
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0015038985 BIOSIS NO.: 200400409774
Serum and glucocorticoid inducible kinases functionally regulate ClC-2 channels
AUTHOR: Palmada Monica; Dieter Michael; Boehmer Christoph; %%%Waldegger%%% %%% Siegfried%%%; %%%Lang Florian%%% (Reprint
AUTHOR ADDRESS: Inst Physiol, Univ Tubingen, Gmelinstr 5, D-72076, Tubingen, Germany**Germany
AUTHOR E-MAIL ADDRESS: florian.lang@uni-tuebingen.de
JOURNAL: Biochemical and Biophysical Research Communications 321 (4): p 1001-1006 September 3, 2004 2004
MEDIUM: print
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/3
DIALOG(R)File 5:Biosis Previews(R)
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0015033115 BIOSIS NO.: 200400403904
Activating mutation of the renal epithelial %%%chloride%%% channel ClC-Kb predisposing to hypertension
AUTHOR: Jeck Nikola; %%%Waldegger Siegfried%%%; Lampert Angelika; Boehmer Christoph; Waldegger Petra; Lang Philipp A; Wissinger Bernd; Friedrich Bjoern; Risler Teut; Moehle Robert; Lang Undine E; Zill Peter; Bondy Brigitta; Schaeffeler Elke; Asante-Poku Stephen; Seyberth Hannsjoerg; Schwab Matthias; %%%Lang Florian%%% (Reprint
AUTHOR ADDRESS: Dept Physiol, Univ Tubingen, Gmelinstr 5, D-72076, Tubingen, Germany**Germany
AUTHOR E-MAIL ADDRESS: florian.lang@uni-tuebingen.de
JOURNAL: Hypertension (Baltimore) 43 (6): p1175-1181 June 2004 2004
MEDIUM: print
ISSN: 0194-911X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/4
DIALOG(R)File 5:Biosis Previews(R)
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0014294469 BIOSIS NO.: 200300253188

Regulation of channels by the serum and glucocorticoid-inducible kinase: Implications for transport, excitability and cell proliferation.
AUTHOR: %%%Lang Florian%%% (Reprint); Henke Guido; Embark Hamdy M; %%%Waldegger Siegfried%%%; Palmada Monica; Bohmer Christoph; Vallon Volker
AUTHOR ADDRESS: Physiologisches Institut I, Gmelinstrasse 5, D-72076, Tuebingen, Germany**Germany
AUTHOR E-MAIL ADDRESS: florian.lang@uni-tuebingen.de
JOURNAL: Cellular Physiology and Biochemistry 13 (1): p41-50 2003 2003
MEDIUM: print
ISSN: 1015-8987
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

7/3/5
DIALOG(R)File 5:Biosis Previews(R)

0013015107 BIOSIS NO.: 200100186946
Cloning and characterization of SLC26A6, a novel member of the solute carrier 26 gene family
AUTHOR: %%%Waldegger Siegfried%%% (Reprint); Moschen Ivano; Ramirez Alfredo ; Smith Richard J H; Ayadi Hammadi; %%%Lang Florian%%%; Kubisch Christian
AUTHOR ADDRESS: Zentrum fuer Kinderheilkunde, Universitaet Marburg, Deutschhausstr. 12, D-35037, Marburg, Germany**Germany
JOURNAL: Genomics 72 (1): p43-50 February 15, 2001 2001
MEDIUM: print
ISSN: 0888-7543
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/6
DIALOG(R)File 5:Biosis Previews(R)

0012052103 BIOSIS NO.: 199900311763
Functional characterization of the betaine/gamma-aminobutyric acid transporter BGT-1 expressed in Xenopus oocytes
AUTHOR: Matskevitch Ioulia; Wagner Carsten A; Stegen Carola; Broeer Stefan; Noll Birgitta; Risler Teut; Kwon H Moo; Handler Joseph S; %%%Waldegger%%% %%% Siegfried%%%; Busch Andreas E; %%%Lang Florian%%% (Reprint
AUTHOR ADDRESS: Institute of Physiology I, University of Tuebingen, Gmelinstrasse 5, 72076, Tuebingen, Germany**Germany
JOURNAL: Journal of Biological Chemistry 274 (24): p16709-16716 June 11, 1999 1999
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/7
DIALOG(R)File 5:Biosis Previews(R)

0010414706 BIOSIS NO.: 199699048766
Expression of a renal type I sodium/phosphate transporter (NaPi-1) induces a conductance in Xenopus oocytes permeable for organic and inorganic anions
AUTHOR: Busch Andreas E (Reprint); Schuster Andreas; %%%Waldegger%%% %%% Siegfried%%%; Wagner Carsten A; Zempel Guenther; Broer Stefan; Biber Juerg; Murer Heini; %%%Lang Florian%%%
AUTHOR ADDRESS: Inst. Physiol. I, Eberhard-Karls-Universitaet Tuebingen, Gmelinstrasse 5, D-72076 Tuebingen, Germany**Germany
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 93 (11): p5347-5351 1996 1996

ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

7/3/8
DIALOG(R)File 5:Biosis Previews(R)
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0010108804 BIOSIS NO.: 199698576637
The type I phosphate (P-i) transporter is a novel anion channel
AUTHOR: Busch Andreas E (Reprint); Schuster Andreas (Reprint); %%%Waldegger%%%
%%% Siegfried%%% (Reprint); Wagner Carsten A (Reprint); Biber Juerg; Murer
Heini; %%%Lang Florian%%% (Reprint
AUTHOR ADDRESS: Univ. Tuebingen, Tuebingen, Germany**Germany
JOURNAL: Journal of the American Society of Nephrology 6 (3): p359 1995
1995
CONFERENCE/MEETING: Annual Meeting of the American Society of Nephrology
San Diego, California, USA November 5-8, 1995; 19951105
ISSN: 1046-6673
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

7/3/9
DIALOG(R)File 5:Biosis Previews(R)
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0009508649 BIOSIS NO.: 199497529934
Molecular basis of I-SK protein regulation by oxidation and chelation
AUTHOR: Busch Andreas E (Reprint); %%%Waldegger Siegfried%%% (Reprint);
Herzer Tobias (Reprint); Raber Gertraud (Reprint); Gulbins Erich
(Reprint); Swanson Richard; Folander Kimberly; Takumi Toru; Moriyoshi
Koki; Nakanishi Shigetada; %%%Lang Florian%%% (Reprint
AUTHOR ADDRESS: Physiol. Inst. I, Univ. Tuebingen, Tuebingen, Germany**
Germany
JOURNAL: Journal of the American Society of Nephrology 5 (3): p283 1994
1994
CONFERENCE/MEETING: Abstracts Submitted for the 27th Annual Meeting of the
American Society of Nephrology Orlando, Florida, USA October 26-29, 1994;
19941026
ISSN: 1046-6673
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

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